

Ref #	Hits	Search Query	DBs	Default Operator	Plurals	Time Stamp
S1	3	US-5789538-\$.DID. OR US-6007408-\$.DID. OR US-6013453-\$.DID.	USPAT	ADJ	ON	2004/07/19 12:41
S2	3	binding site with cellular chromatin	USPAT	ADJ	ON	2004/07/17 15:48
S3	3	S2 not S1	USPAT	ADJ	ON	2004/07/17 15:48
S4	3	US-5306619-\$.DID. OR US-6410248-\$.DID. OR US-6007988-\$.DID.	USPAT	ADJ	ON	2004/07/19 12:44
S5	3	US-5789538-\$.DID. OR US-6007408-\$.DID. OR US-6013453-\$.DID.	USPAT	ADJ	ON	2004/07/19 12:50
S6	23554	chromatin or chromosome or episome or nucleosome	USPAT	ADJ	ON	2004/07/19 12:49
S7	274	S6 with binding site	USPAT	ADJ	ON	2004/07/19 12:49
S8	2	S7 WITH (zinc finger)	USPAT	ADJ	ON	2004/07/19 12:52
S9	90	S7 WITH (protein)	USPAT	ADJ	ON	2004/07/19 12:52
S10	480	bind\$ with minor groove	USPAT	ADJ	ON	2004/07/19 13:00
S11	71	S10 with protein	USPAT	ADJ	ON	2004/07/19 13:00
S12	71	S11 not S9	USPAT	ADJ	ON	2004/07/19 13:01
S13	1	S12 with chromatin	USPAT	ADJ	ON	2004/07/19 13:02
S14	10	S10 with zinc finger	USPAT	ADJ	ON	2004/07/19 13:02

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FILE 'HOME' ENTERED AT 13:36:35 ON 19 JUL 2004

=> index biosci

FILE 'DRUGMONOG' ACCESS NOT AUTHORIZED

COST IN U.S. DOLLARS

	SINCE FILE	TOTAL
	ENTRY	SESSION
FULL ESTIMATED COST	0.21	0.21

INDEX 'ADISCTI', ADISINSIGHT, ADISNEWS, AGRICOLA, ANABSTR, AQUASCI, BIOBUSINESS,
BIOCOMMERCE, BIOSIS, BIOTECHABS, BIOTECHDS, BIOTECHNO, CABA, CANCERLIT,
CAPLUS, CEABA-VTB, CIN, CONFSCI, CROFB, CROFU, DISSABS, DDFB, DDFU,
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70 FILES IN THE FILE LIST IN STNINDEX

Enter SET DETAIL ON to see search term postings or to view
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=> s chromatatin (p) bind?

2 FILE ADISCTI
1 FILE ADISINSIGHT
1* FILE ADISNEWS
247 FILE AGRICOLA
1 FILE ANABSTR
81 FILE AQUASCI
10 FILE BIOBUSINESS
0* FILE BIOTECHABS
6287 FILE BIOSIS
127* FILE BIOTECHDS
127* FILE BIOTECHNO
3979* FILE CABA
405 FILE CANCERLIT
1956 FILE CAPLUS
7229 FILE CEABA-VTB
3* FILE CIN
53 FILE CONFSCI
5 FILE CROFB
3 FILE CROFU
800 FILE DISSABS
107 FILE DDFB
91 FILE DDFU
1329 FILE DGENE
25 FILES SEARCHED...
107 FILE DRUGB
2 FILE INSDRUGNEWS
204 FILE DRUGU
2 FILE INSRSEARCH

128 FILE EMBAL
5214 FILE ENGASE
4341* FILE ESHIOBASE
575* FILE FEDRIP
0* FILE FORAD
0* FILE FORSE
2* FILE FROSTI
5* FILE FSTA
464 FILE GENBANK
6 FILE HEALSAFE
110 FILE IPAT
102 FILE JICST-EPJUS
3* FILE KOSMET
3424 FILE LIFESCI
0* FILE MEDICINF
5940 FILE MEDLINE
60 FILE NIOSHTIC
70* FILE NTIS
0* FILE NUTRACUT
2 FILE OCEAN
1051* FILE PASCAL
0* FILE PHARMAML
1 FILE PHIN
24 FILE PROMT
58 FILES SEARCHED...
5900 FILE SCISEARCH
2647 FILE TOXCENTER
1414 FILE USPATFOLL
62 FILE USPAT2
1 FILE VETU
107 FILE WPIDS
2 FILE WPIFV
107 FILE WPINDEX

54 FILES HAVE ONE OR MORE ANSWERS, 70 FILES SEARCHED IN STNINDEX

L1 QUE CHROMATIN (P) BIND?

=> file biotechno biosis lifesci medline
COST IN U.S. DOLLARS

	SINCE FILE	TOTAL
	ENTRY	SESSION
FULL ESTIMATED COST	2.28	2.49

FILE 'BIOTECHNO' ENTERED AT 13:38:58 ON 19 JUL 2004

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FILE 'BIOSIS' ENTERED AT 13:38:58 ON 19 JUL 2004

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FILE 'MEDLINE' ENTERED AT 13:38:58 ON 19 JUL 2004

=> s 11

PROXIMITY OPERATOR LEVEL NOT CONSISTENT WITH
FIELD CODE - 'AND' OPERATOR ASSUMED 'CHROMATIN (P) BIND?'

L2 19630 L1

=> s 12 and (zinc (w) finger) or zfp

L3 784 L2 AND (ZINC (W) FINGER) OR ZFP

=> s 13 and intracell?

L4 14 L3 AND INTRACELL?

=> d 14 bib ab 1-14

L4 ANSWER 1 OF 14 BIOTECHNO COPYRIGHT 2004 Elsevier Science B.V. on STN

AN 2003:37272164 BIOTECHNO

TI The rise of DNA methylation and the importance of ***chromatin*** on

LA multidrug resistance in cancer

CS Baker E.K.; El-Osta A.

Vict. 3181, Australia.

E-mail: assam.el-osta@baker.edu.au

SO Experimental Cell Research, (01 NOV 2003), 290/2 (177-194), 197

DT references(s)

CY CODEN: ECREAL ISSN: 0014-4827

LA Journal; General Review

SL United States

AB English

In recent years, the different classes of drugs and regimens used clinically have provided an improvement in tumour management. However, treatment is often palliative for the majority of cancer patients. Transformed cells respond poorly to chemotherapy mainly due to the development of the multidrug resistance (MDR) phenotype. Response to treatment does not generally result in complete remission and disease cure is uncommon for patients presenting with advanced stage cancer. Successful treatment of cancer requires a clearer understanding of chemotherapeutic resistance. Here, we examine what is known of one of the most extensively studied mechanisms of cellular drug resistance. The human multidrug resistance gene 1 (MDR1) is associated with expression of p-glycoprotein (Pgp). A transmembrane protein, Pgp acts as an efflux pump and reduces ***intracellular*** drug levels and thus its effectiveness as an antitumor agent. The precise mechanism of transcriptional regulation has been unclear due to the complex regulatory nature of the gene. It has become increasingly apparent that trans-activation or genetic amplification is by no means the only mechanism of activation. Consequently, alternative pathways have received more attention in the area of epigenetics to help explain transcriptional competence at a higher level of organization. The goal of this article is to highlight important findings in the field of methylation and explain how they impinge on MDR1 gene regulation. In this review, we cover the current information and postulate that epigenetic modification of MDR1 ***chromatin*** influences gene transcription in leukaemia. Finally, we explore transcriptional regulation and highlight recent progress with engineered ***ZFP*** 's (***zinc*** ***finger*** proteins). .COPYRG. 2003 Elsevier Inc. All rights reserved.

L4 ANSWER 2 OF 14 BIOTECHNO COPYRIGHT 2004 Elsevier Science B.V. on STN

AN 2002:34429757 BIOTECHNO

TI Nitrate and the GATA factor Area are necessary for in vivo

LA ***binding*** of NirA, the pathway-specific transcriptional activator of Aspergillus nidulans

CS Narendia F.; Goller S.P.; Wolschek M.; Strauss J.

Vienna, Austria.

E-mail: jstrauss@edvz.boku.ac.at

SO Molecular Microbiology, (2002), 44/2 (573-583), 53 reference(s)

DT CODEN: MOMIEE ISSN: 0950-382X

CY Journal; Article

LA United Kingdom

SL English

AB In Aspergillus nidulans, the genes coding for nitrate reductase (niaD) and nitrite reductase (niaI), are transcribed divergently from a common promoter region of 1200 basepairs. We have previously characterized the relevant cis-acting elements for the two synergistically acting transcriptional activators NirA and Area. We have further shown that Area is constitutively bound to a central cluster of four GATA sites, and is involved in opening the ***chromatin*** structure over the promoter region thus making additional cis-acting ***binding*** sites accessible. Here we show that the asymmetric mode of NirA-DNA interaction determined in vitro is also found in vivo. ***Binding*** of the NirA transactivator is not constitutive as in other binuclear C6-Zn.sup.2+.sup.+cluster proteins but depends on nitrate induction and, additionally, on the presence of a wild-type area allele. Dissecting the role of Area further, we found that it is required for ***intracellular*** nitrate accumulation and therefore could indirectly exert its effect on NirA via inducer exclusion. We have tested this possibility in a strain accumulating nitrate in the absence of area. We found that in such a strain the ***intracellular*** presence of inducer is not sufficient to promote either ***chromatin*** rearrangement or NirA ***binding***, implying that both processes are directly dependent on Area.

L4 ANSWER 3 OF 14 BIOTECHNO COPYRIGHT 2004 Elsevier Science B.V. on STN

AN 1998:28240528 BIOTECHNO

TI XRCC1 is specifically associated with poly(ADP-ribose) polymerase and negatively regulates its activity following DNA damage

AU Masson M.; Niedergang C.; Schreiber V.; Muller S.; Menissier-De Murcia J.; De Murcia G.

CS G. De Murcia, Ecole Sup. Biotechnol. de Strasbourg, UPR 9003 du Ctr. Natl. Rech. Sci., Boulevard S. Brant, F-67400 Illkirch-Graffenstaden, France.

E-mail: demurcia@esbs.u-strasbg.fr

SO Molecular and Cellular Biology, (1998), 18/6 (3563-3571), 58 reference(s)

DT CODEN: MCEBDA ISSN: 0270-7306

CY Journal; Article

LA United States

SL English

AB Poly(ADP-ribose) polymerase (PARP; EC 2.4.2.30) is a ***zinc*** ***finger*** DNA- ***binding*** protein that detects and signals DNA strand breaks generated directly or indirectly by genotoxic agents. In response to these breaks, the immediate poly(ADP-ribose)ylation of nuclear proteins involved in ***chromatin*** architecture and DNA

metabolism converts DNA damage into ***intracellular*** signals that can activate DNA repair programs or cell death options. To have greater insight into the physiological function of this enzyme, we have used the two-hybrid system to find genes encoding proteins putatively interacting with PARP. We have identified a physical association between PARP and the base excision repair (BER) protein XRCC1 (X-ray repair cross-complementing 1) in the Saccharomyces cerevisiae system, which was further confirmed to exist in mammalian cells. XRCC1 interacts with PARP by its central region (amino acids 301 to 402), which contains a BRCT (BRCA1 C terminus) module, a widespread motif in DNA repair and DNA damage-responsive cell cycle checkpoint proteins. Overexpression of XRCC1 in Cos-7 or HeLa cells dramatically decreases PARP activity *in vivo*, reinforcing the potential protective function of PARP at DNA breaks. Given that XRCC1 is also associated with DNA ligase III via a second BRCT module and with DNA polymerase β , our results provide strong evidence that PARP is a member of a BER multiprotein complex involved in the detection of DNA interruptions and possibly in the recruitment of XRCC1 and its partners for efficient processing of these breaks in a coordinated manner. The modular organizations of these interactors, associated with small conserved domains, may contribute to increasing the efficiency of the overall pathway.

L4 ANSWER 4 OF 14 BIOSIS COPYRIGHT 2004 BIOLOGICAL ABSTRACTS INC. on STN
AN 2004:13460 BIOSIS
DN PREV200400017673

TI The rise of DNA methylation and the importance of chromatin on multidrug resistance in cancer.

AU Baker, Emma K.; El-Osta, Assem [Reprint Author]
CS Alfred Medical Research and Education Precinct (AMREP), Baker Medical Research Institute, Epigenetics in Human Health and Disease Laboratory, Commercial Road, Second Floor, Prahran, VIC, 3181, Australia
assem.el-osta@baker.edu.au

SO Experimental Cell Research, (November 1 2003) Vol. 290, No. 2, pp. 177-194. Print.

ISSN: 0014-4827 (ISSN print).

Article

General Review; (Literature Review)

English

Entered STN: 24 Dec 2003

Last Updated on STN: 24 Dec 2003

In recent years, the different classes of drugs and regimens used clinically have provided an improvement in tumour management. However, treatment is often palliative for the majority of cancer patients. Transformed cells respond poorly to chemotherapy mainly due to the development of the multidrug resistance (MDR) phenotype. Response to treatment does not generally result in complete remission and disease cure is uncommon for patients presenting with advanced stage cancer. Successful treatment of cancer requires a clearer understanding of chemotherapeutic resistance. Here, we examine what is known of one of the most extensively studied mechanisms of cellular drug resistance. The human multidrug resistance gene 1 (MDR1) is associated with expression of p-glycoprotein (Pgp). A transmembrane protein, Pgp acts as an efflux pump and reduces ***intracellular*** drug levels and thus its effectiveness as an antitumor agent. The precise mechanism of transcriptional regulation has become unclear due to the complex regulatory nature of the gene. It has become increasingly apparent that trans-activation or genetic amplification is by no means the only mechanism of activation.

Consequently, alternative pathways have received more attention in the area of epigenetics to help explain transcriptional competence at a higher level of organization. The goal of this article is to highlight important findings in the field of methylation and explain how they impinge on MDR1 gene regulation. In this review, we cover the current information and postulate that epigenetic modification of MDR1 chromatin influences gene transcription in leukaemia. Finally, we explore transcriptional regulation and highlight recent progress with engineered ***ZFP***'s (zinc finger proteins).

L4 ANSWER 5 OF 14 BIOSIS COPYRIGHT 2004 BIOLOGICAL ABSTRACTS INC. on STN
AN 2002:174793 BIOSIS
DN PREV200200174793

TI Retrovirally expressed metal response element-binding transcription factor-1 normalizes metallothionein-I gene expression and protects cells against zinc, but not cadmium, toxicity.

AU Solis, Willy A.; Childs, Nicole L.; Weedon, Michael N.; He, Lei; Nebert, Daniel W.; Dalton, Timothy P. [Reprint author]
CS Department of Environmental Health, University of Cincinnati Medical Center, Cincinnati, OH, 45267-0056, USA
tim.dalton@uc.edu

SO Toxicology and Applied Pharmacology, (January 15, 2002) Vol. 178, No. 2, pp. 93-101. Print.

CODEN: TXAP9. ISSN: 0041-008X.

Article

English

Entered STN: 6 Mar 2002

Last Updated on STN: 6 Mar 2002

Metal response element (MRE) transcription factor-1 (MTF1), a member of the Cys2-His2 class of ***zinc*** - ***finger*** transcription factors, is best known for its robust transcriptional regulation of mammalian metallothionein (MT) genes. MTF1 is also believed to play a generalized role in regulating genes involved in protection against heavy metals and oxidative stress. MTF1 ***binding*** to MRE motifs is regulated by changes in ***intracellular*** zinc (Zn²⁺) concentration. Molecular dissection of MTF1 has been hindered by its high constitutive trans-activity following transient transfection and the failure of these systems to examine genes packaged in native ***chromatin***. In developing a system to avoid these problems, we employed a high-efficiency retroviral transduction system to reintroduce MTF1 into mouse MTF1(-/-) knockout cells (dko7). Electrophoretic mobility shift assays demonstrated that MTF1 retrovirally transduced dko7 cells (MTF1dko7) possess levels of inducible MTF1-MRE ***binding*** activity similar to that seen in mouse hepatoma Hepa-1 cells, and MTF1 ***binding*** could be modulated over a 20-fold range by varying the concentration of Zn²⁺ present in the culture medium. The dko7 cells exhibited no change in M1 gene expression upon Zn²⁺ or cadmium (Cd²⁺) treatment; in contrast, in MTF1dko7 cells, Zn²⁺ or Cd²⁺ induced MTF1 mRNA accumulation in a dose-dependent manner. Interestingly, MTF1dko7 cells showed resistance to Zn²⁺ toxicity, but negligible resistance to Cd²⁺. Concomitantly, MTF1 protein levels in MTF1dko7 cells were inducible to the same degree as that in Hepa-1 cells when treated with Zn²⁺, but not with Cd²⁺. Together, our studies suggest that MTF1-mediated regulation of gene expression is sufficient to protect cells against Zn²⁺ toxicity and may be necessary but not sufficient to protect cells against Cd²⁺ toxicity.

L4 ANSWER 6 OF 14 BIOSIS COPYRIGHT 2004 BIOLOGICAL ABSTRACTS INC. on STN

AN 1998:296836 BIOSIS
 DN PREV199800296836
 TI XRCC1 is specifically associated with poly(ADP-ribose) polymerase and negatively regulates its activity following DNA damage.
 AU Masson, Murielle; Niedergang, Claude; Schreiber, Valerie; Muller, Sylviane; Menissier-De Murcia, Josiane; De Murcia, Gilbert [Reprint author]
 CS Ecole Supérieure Biotechnol., Strasbourg, UPR 9003 Cont. Natl. Rech. Sci., Boulevard S. Brant, F-67400 Illkirch-Graffenstaden, France
 SO Molecular and Cellular Biology, (June, 1998) Vol. 18, No. 6, pp. 3563-3571. print.
 CODEN: MCEBD4. ISSN: 0270-7306.
 DT Article
 LA English
 ED Entered STN: 15 Jul 1998
 AB Last Updated on STN: 15 Jul 1998
 Poly(ADP-ribose) polymerase (PARP; EC 2.4.2.30) is a ***zinc*** - ***finger*** DNA- ***binding*** protein that detects and signals DNA strand breaks generated directly or indirectly by genotoxic agents. In response to these breaks, the immediate poly(ADP-ribose)ylation of nuclear proteins involved in ***chromatin*** architecture and DNA metabolism converts DNA damage into ***intracellular*** signals that can activate DNA repair programs or cell death options. To have greater insight into the physiological function of this enzyme, we have used the two-hybrid system to find genes encoding proteins putatively interacting with PARP. We have identified a physical association between PARP and the base excision repair (BER) protein XRCC1 (X-ray repair cross-complementing 1) in the Saccharomyces cerevisiae system, which was further confirmed to exist in mammalian cells. XRCC1 interacts with PARP by its central region (amino acids 301 to 402), which contains a BRCT (BRCA1 C terminus) module, a widespread motif in DNA repair and DNA damage-responsive cell cycle checkpoint proteins. Overexpression of XRCC1 in Cos-7 or HeLa cells dramatically decreases PAR-P activity in vivo, reinforcing the potential protective function of PARP at DNA breaks. Given that XRCC1 is also associated with DNA ligase III via a second BRCT module and with DNA polymerase beta, our results provide strong evidence that PARP is a member of a BER multiprotein complex involved in the detection of DNA interruptions and possibly in the recruitment of XRCC1 and its partners for efficient processing of these breaks in a coordinated manner. The modular organizations of these interactors, associated with small conserved domains, may contribute to increasing the efficiency of the overall pathway.

SL English
 AB Small molecule inhibitors of human immunodeficiency virus, type 1 (HIV-1) have been extremely successful but are associated with a myriad of undesirable effects and require lifelong daily dosing. In this study we explore an alternative approach, that of inducing ***intracellular*** immunity using designed, ***zinc*** - ***finger*** -based transcription factors. Three transcriptional repression proteins were engineered to ***bind*** sites in the HIV-1 promoter that were expected to be both accessible in ***chromatin*** structure and highly conserved in sequence structure among the various HIV-1 subgroups. Transient transfection assays identified one factor, KRAB-HLTR3, as being able to achieve 100-fold repression of an HIV-1 promoter. Specificity of repression was demonstrated by the lack of repression of other promoters. This factor was further shown to repress the replication of several HIV-1 viral strains 10- to 100-fold in T-cell lines and primary human peripheral blood mononuclear cells. Repression was observed for at least 18 days with no significant cytotoxicity. Stable T-cell lines expressing the factor also do not show obvious signs of cytotoxicity. These characteristics present KRAB-HLTR3 as an attractive candidate for development in an ***intracellular*** immunization strategy for anti-HIV-1 therapy.

L4 ANSWER 8 OF 14 LIFESCI COPYRIGHT 2004 CSA on STN
 AN 2002:44734 LIFESCI
 TI Retrovirally Expressed Metal Response Element-Binding Transcription Factor-1 Normalizes Metallothionein-1 Gene Expression and Protects Cells against Zinc, but Not Cadmium, Toxicity
 AU Soils, W.A.; Childs, N.L.; Weedon, M.N.; He, L.; Nebert, D.W.; Dalton, T.P.*
 CS Center for Environmental Genetics, Department of Environmental Health, University of Cincinnati Medical Center, Cincinnati, Ohio, 45267-0056; E-mail: tim.dalton@uc.edu
 SO Toxicology and Applied Pharmacology [Toxicol. Appl. Pharmacol.], (20020115) vol. 178, no. 2, pp. 93-101.
 ISSN: 0041-008X.
 DT X
 FS X
 LA English
 SL English
 AB Metal response element (MRE) transcription factor-1 (MTF1), a member of the Cys sub(2)-His sub(2) class of ***zinc*** - ***finger*** transcription factors, is best known for its robust transcriptional regulation of mammalian metallothionein (MT) genes. MTF1 is also believed to play a generalized role in regulating genes involved in protection against heavy metals and oxidative stress. MTF1 ***binding*** to MRE motifs is regulated by changes in ***intracellular*** zinc (Zn super(2+)) concentration. Molecular dissection of MTF1 has been hindered by its high constitutive trans-activity following transient transfection and the failure of these systems to examine genes packaged in native ***chromatin***. In developing a system to avoid these problems, we employed a high-efficiency retroviral transduction system to reintroduce MTF1 into mouse Mtf1(-/-) knockout cells (dko7). Electrophoretic mobility shift assays demonstrated that MTF1 retrovirally transduced dko7 cells (MTF1dko7) possess levels of inducible MTF1-MRE ***binding*** activity similar to that seen in mouse hepatoma Hepa-1 cells, and MTF1 ***binding*** could be modulated over a 20-fold range by varying the concentration of Zn super(2+) present in the culture medium. The dko7 cells exhibited no change in Mtf1 gene expression upon Zn super(2+) or

cadmium (Cd super(2+)) treatment; in contrast, in MTF1cdk7 cells, Zn super(2+) or Cd super(2+) induced MTF1 mRNA accumulation in a dose-dependent manner. Interestingly, MTF1cdk7 cells showed resistance to Zn super(2+) toxicity, but negligible resistance to Cd super(2+). Concomitantly, MTF1 protein levels in MTF1cdk7 cells were inducible to the same degree as that in Hepa-1 cells when treated with Zn super(2+), but not with Cd super(2+). Together, our studies suggest that MTF1-mediated regulation of gene expression is sufficient to protect cells against Zn super(2+) toxicity and may be necessary but not sufficient to protect cells against Cd super(2+) toxicity. [copy] 2002 Elsevier Science (USA).

ANSWER 9 OF 14 LIFESCI COPYRIGHT 2004 CSA on STN
 1998:107039 LIFESCI
 TI XRCOI is specifically associated with poly(ADP-ribose) polymerase and negatively regulates its activity following DNA damage
 AU Masson, M.; Niedergang, C.; Schreiber, V.; Muller, S.; Menissier-de Murcia, J.; De Murcia, G.
 CS Ecole Supérieure de Biotechnologie de Strasbourg, UPR 9003 du Centre National de la Recherche Scientifique, Boulevard S. Brant, F-67400 Illkirch-Graffenstaden, France
 SO Mol. Cell. Biol., (19980600) vol. 18, no. 6, pp. 3563-3571.
 ISSN: 0270-7306.
 Journal
 DT N
 FS N
 LA English
 SL English
 AB Poly(ADP-ribose) polymerase (PARP; EC 2.4.2.30) is a ***zinc*** - ***finger*** DNA- ***binding*** protein that detects and signals DNA strand breaks generated directly or indirectly by genotoxic agents. In response to these breaks, the immediate poly(ADP-ribose)ylation of nuclear proteins involved in ***chromatin*** architecture and DNA metabolism converts DNA damage into ***intracellular*** signals that can activate DNA repair programs or cell death options. To have greater insight into the physiological function of this enzyme, we have used the two-hybrid system to find genes encoding proteins putatively interacting with PARP. We have identified a physical association between PARP and the base excision repair (BER) protein XRCOI (X-ray repair cross-complementing 1) in the *Saccharomyces cerevisiae* system, which was further confirmed to exist in mammalian cells. XRCOI interacts with PARP by its central region (amino acids 301 to 402), which contains a BRCT (BRCA1 C terminus) module, a widespread motif in DNA repair and DNA damage-responsive cell cycle checkpoint proteins. Overexpression of XRCOI in Cos-7 or HeLa cells dramatically decreases PARP activity *in vivo*, reinforcing the potential protective function of PARP at DNA breaks. Given that XRCOI is also associated with DNA ligase III via a second BRCT module and with DNA polymerase beta, our results provide strong evidence that PARP is a member of a BER multiprotein complex involved in the detection of DNA interruptions and possibly in the recruitment of XRCOI and its partners for efficient processing of these breaks in a coordinated manner. The modular organization of these interactors, associated with small conserved domains, may contribute to increasing the efficiency of the overall pathway.

ANSWER 10 OF 14 MEDLINE on STN
 2004:172762 MEDLINE
 PubMed ID: 14734553
 TI Attenuation of HIV-1 replication in primary human cells with a designed

zinc ***finger*** transcription factor.
 Segal David J; Goncalves Joao; Eberhardt Scott; Swan Christina H; Torbett Bruce E; Li Xuelin; Barbas Carlos F 3rd
 CS The Skaggs Institute for Chemical Biology and the Departments of Molecular Biology and Chemistry, The Scripps Research Institute, La Jolla, California 92037, USA.
 NC GM065059 (NIGMS)
 R01 AI49165 (NIHID)
 SO Journal of biological chemistry, (2004 Apr 9) 279 (15) 14509-19.
 Journal code: 2985121R. ISSN: 0021-9258.
 CY United States
 DT Journal; Article; (JOURNAL ARTICLE)
 LA English
 FS Priority Journals
 OS GENBANK:AY518586; GENBANK:AY518587; GENBANK:AY518588
 EM 200406
 ED Entered STN: 20040407
 Last Updated on STN: 20040602
 Entered Medline: 20040601
 AB Small molecule inhibitors of human immunodeficiency virus, type 1 (HIV-1) have been extremely successful but are associated with a myriad of undesirable effects and require lifelong daily dosing. In this study we explore an alternative approach, that of inducing ***intracellular*** immunity using designed, ***zinc*** ***finger*** -based transcription factors. Three transcriptional repression proteins were engineered to ***bind*** sites in the HIV-1 promoter that were expected to be both accessible in ***chromatin*** structure and highly conserved in sequence structure among the various HIV-1 subgroups. Transient transfection assays identified one factor, KRAB-HTR3, as being able to achieve 100-fold repression of an HIV-1 promoter. Specificity of repression was demonstrated by the lack of repression of other promoters. This factor was further shown to repress the replication of several HIV-1 viral strains 10- to 100-fold in T-cell lines and primary human peripheral blood mononuclear cells. Repression was observed for at least 18 days with no significant cytotoxicity. Stable T-cell lines expressing the factor also do not show obvious signs of cytotoxicity. These characteristics present KRAB-HTR3 as an attractive candidate for development in an ***intracellular*** immunization strategy for anti-HIV-1 therapy.

ANSWER 11 OF 14 MEDLINE on STN
 2003:491182 MEDLINE
 DN PubMed ID: 14567978
 TI The rise of DNA methylation and the importance of chromatin on multidrug resistance in cancer.
 AU Baker Emma K; El-Osta Assam
 CS The Alfred Medical Research and Education Precinct, Baker Medical Research Institute, Epigenetics in Human Health and Disease Laboratory, Second Floor, Commercial Road, Prahran, Victoria 3181, Australia.
 SO Experimental cell research, (2003 Nov 1) 290 (2) 177-94. Ref: 197
 Journal code: 03733226. ISSN: 0014-4827.
 CY United States
 DT Journal; Article; (JOURNAL ARTICLE)
 LA English
 FS Priority Journals
 General Review; (REVIEW)
 (REVIEW, ACADEMIC)

EM	200312
ED	Entered

in recent years, the different classes of drugs and regimens used clinically have provided an improvement in tumour management. However, treatment is often palliative for the majority of cancer patients. Transformed cells respond poorly to chemotherapy mainly due to the development of the multidrug resistance (MDR) phenotype. Response to treatment does not generally result in complete remission and disease cure is uncommon for patients presenting with advanced stage cancer. Successful treatment of cancer requires a clearer understanding of chemotherapeutic resistance. Here, we examine what is known of one of the most extensively studied mechanisms of cellular drug resistance. The human multidrug resistance gene 1 (MDR1) is associated with expression of p-glycoprotein (Pgp). A transmembrane protein, Pgp acts as an efflux pump and reduces *****intracellular***** drug levels and thus its effectiveness as an antitumor agent. The precise mechanism of transcriptional regulation has been unclear due to the complex regulatory nature of the gene. It has become increasingly apparent that trans-activation of genetic amplification is by no means the only mechanism of activation. Consequently, alternative pathways have received more attention in the area of epigenetics to help explain transcriptional competence at a higher level of organization. The goal of this article is to highlight important findings in the field of methylation and explain how they impinge on MDR1 gene regulation. In this review, we cover the current information and postulate that epigenetic modification of MDR1 chromatin influences gene transcription in leukaemia. Finally, we explore transcriptional regulation and highlight recent progress with engineered *****2Fp***** 's' (zinc finger proteins).

LL4 ANSWER 12 OF 14 MEDLINE on STN
AN 2002120545 MEDLINE

Retrovirally expressed metal response element-binding transcription factor-1 normalizes metallothionein-1 gene expression and protects cells against zinc, but not cadmium, toxicity.

AU Solis Willy A; Childs Nicole L; Weeden Michael N; He Lei; Nebert Daniel W;
 Dalton Timothy P
 UCS Center for Environmental Genetics, University of Cincinnati Medical
 Center, Cincinnati, Ohio 45267-0056, USA.

NC
P30 ES06096 (NIEHS)
R01 AG09235 (NIA)
R01 ES10416 (NIEHS)
Toxicology and appli
Journal code: 041657

United States
Journal; Article; (JOURNAL ARTICLE)
English
Priority Journals
200203

AB Entered medicine: 20020307
Metal response element (MRE) transcription factor-1 (MTF1), a member of the Cys2-His2 class of ***zinc*** - **finger** transcription factors, is best known for its robust transcriptional regulation of

mammalian metallothionein (MT) genes. MTf1 is also believed to play a generalized role in regulating genes involved in protection against heavy metals and oxidative stress. MTf1 **binding** to MRE motifs is regulated by changes in **intracellular** zinc (Zn(2+)) concentration. Molecular dissection of MTf1 has been hindered by its high constitutive trans-activity following transient transfection and the failure of these systems to examine genes packaged in native **chromatin**... In developing a system to avoid these problems, we employed a high-efficiency retroviral transduction system to reintroduce MTf1 into mouse Mtfl1(-/-) knockout cells (dko7). Electrophoretic mobility shift assays demonstrated that MTf1 retrovirally transduced dko7 cells (MTf1dko7) possess levels of inducible MTf1-MRE **binding** activity similar to that seen in mouse hepatoma Hepa-1 cells, and MTf1 **binding** could be modulated over a 20-fold range by varying the concentration of Zn(2+) present in the culture medium. The dko7 cells exhibited no change in Mt1 gene expression upon Zn(2+) or cadmium (Cd(2+)) treatment; in contrast, in MTf1dko7 cells, Zn(2+) or Cd(2+) induced MTf1 mRNA accumulation in a dose-dependent manner. Interestingly, MTf1dko7 cells showed resistance to Zn(2+) toxicity, but negligible resistance to Cd(2+). Concomitantly, Mt1 protein levels in MTf1dko7 cells were inducible to the same degree as that in Hepa-1 cells when treated with Zn(2+), but not with Cd(2+). Together, our studies suggest that MTf1-mediated regulation of gene expression is sufficient to protect cells against Zn(2+) toxicity and may be necessary but not sufficient to protect cells against Cd(2+) toxicity.

2002 Elsevier Science (USA).

2002 Elsevier Science (USA).

L4 ANSWER 13 OF 14 MEDLINE on STN
AN 2001539383 MEDLINE
DN PubMed ID: 11586467
TI Transcriptional regulation in hepatic stellate cells.

CS Division of Liver Diseases, Department of Medicine, Mount Sinai School of Medicine, 1425 Madison Ave., New York, NY 10029, USA.
 SC Seminars in liver disease, (2001 Aug) 21 (3) 395-95. Ref: 81
 Journal code: 8110297. ISSN: 0272-8087.

CY United States
 DT Journal; Article; (JOURNAL ARTICLE)

General Review; (REVIEW)
(REVIEW, TUTORIAL)

LA English
FS Priority Journals
EM 200111

AB Modulation of gene expression through altered transcription regulation in stellate cell behavior in normal liver and following hepatic injury. Transcription factors are generally classified according to conserved motifs within either the activation- or DNA- binding domains of the molecules. Transcriptional activity in stellate cells represents a delicate fine tuning of multiple inputs. Activities of these transcription factors are modified by their intracellular localization, rate and pathway of degradation, oligomerization, and interactions with heterologous factors and chromatin, as well as by posttranslational modifications, including phosphorylation, glycosylation, and acetylation. General paradigms of transcriptional

control are increasingly being validated in hepatic stellate cells, particularly involving the transcription factors CCAAT/enhancer-binding proteins, c-myc, CREB, nuclear factor kappaB, peroxisome proliferator-activated receptor, and Kruppel-like zinc factors. Although there are no simple rules that govern mechanisms of transcriptional regulation in stellate cells, continued advances will yield new insights into their role in normal liver homeostasis and in the response to injury.

ANSWER 14 OF 14 MEDLINE on STN
 AN 1998252943 MEDLINE
 DN PubMed ID: 9584196
 TI XRCC1 is specifically associated with poly(ADP-ribose) polymerase and negatively regulates its activity following DNA damage.
 AU Masson M; Niedergang C; Schreiber V; Muller S; Menissier-de Murcia J; de Murcia G
 CS UPR 9003 du Centre National de la Recherche Scientifique, Cancerogenese et Mutagenese Moleculaire et Structurale, Ecole Superieure de Biotechnologie de Strasbourg, 67400 Illkirch-Graffenstaden, France.
 SO Molecular and cellular biology. (1998 Jun) 18 (6) 3563-71.
 JOURNAL CODE: 8105087. ISSN: 0270-7306.
 CY United States
 DT Journal; Article; (JOURNAL ARTICLE)
 LA English
 FS Priority Journals
 EM 199806
 ED Entered STN: 19980625
 Last Updated on STN: 19980625
 Entered Medline: 19980617
 AB Poly(ADP-ribose) polymerase (PARP; EC 2.4.2.30) is a zinc finger protein that detects and signals DNA strand breaks generated directly or indirectly by genotoxic agents. In response to these breaks, the immediate poly(ADP-ribose)ylation of nuclear proteins involved in chromatin architecture and DNA metabolism converts DNA damage into intracellular signals that can activate DNA repair programs or cell death options. To have greater insight into the physiological function of this enzyme, we have used the two-hybrid system to find genes encoding proteins putatively interacting with PARP. We have identified a physical association between PARP and the base excision repair (BER) protein XRCC1 (X-ray repair cross-complementing 1) in the Saccharomyces cerevisiae system, which was further confirmed to exist in mammalian cells. XRCC1 interacts with PARP by its central region (amino acids 301 to 402), which contains a BRCT (BRCA1 C terminus) module, a widespread motif in DNA repair and DNA damage-responsive cell cycle checkpoint proteins. Overexpression of XRCC1 in Cos-7 or HeLa cells dramatically decreases PARP activity in vivo, reinforcing the potential protective function of PARP at DNA breaks. Given that XRCC1 is also associated with DNA ligase III via a second BRCT module and with DNA polymerase beta, our results provide strong evidence that PARP is a member of a BER multiprotein complex involved in the detection of DNA interruptions and possibly in the recruitment of XRCC1 and its partners for efficient processing of these breaks in a coordinated manner. The modular organizations of these interactors, associated with small conserved domains, may contribute to increasing the efficiency of the overall pathway.

=> log h
 COST IN U.S. DOLLARS
 ENTRY 22.10
 FULL ESTIMATED COST 24.59

SESSION WILL BE HELD FOR 60 MINUTES
 STN INTERNATIONAL SESSION SUSPENDED AT 13:40:05 ON 19 JUL 2004
 Connecting via Winsock to STN

Welcome to STN International! Enter x:x

LOGINID:SSSFT7A1653RAW

PASSWORD:
 ***** RECONNECTED TO STN INTERNATIONAL *****
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 FILE 'MEDLINE' ENTERED AT 13:44:54 ON 19 JUL 2004

COST IN U.S. DOLLARS
 ENTRY 22.10
 FULL ESTIMATED COST 24.59

=> s chromatin and binding (w) site and protein
 L5 1608 CHROMATIN AND BINDING (W) SITE AND PROTEIN

=> dup rem l5

PROCESSING IS APPROXIMATELY 75% COMPLETE FOR L5
 PROCESSING COMPLETED FOR L5

L6 842 DUP REM L5 (766 DUPLICATES REMOVED)

=> s l6 and zinc (w) finger

L7 39 L6 AND ZINC (W) FINGER

=> d his

(FILE 'HOME' ENTERED AT 13:36:35 ON 19 JUL 2004)

INDEX 'ADISCTI, ADISINSIGHT, ADISNEWS, AGRICOLA, ANABSTR, AQUASCI, BIOBUSINESS, BIOCOMMERCS, BIOSIS, BIOTECHABS, BIOTECHNO, CABA, CANCERLIT, CAPLUS, CABA-VTB, CIN, CONFSCI, CROFB, DISSABS, DDFB, DDFU, DGENE, DRUGS, DRUGMONOG2, ...' ENTERED AT 13:36:48 ON 19 JUL 2004

SEA CHROMATIN (P) BIND?

 2 FILE ADISCTI
 1 FILE ADISINSIGHT

1* FILE ADISNEWS
247 FILE AGRICOLA
1 FILE ANABSTR
81 FILE AQUASCI
10 FILE BIABUSINESS
0* FILE BIOCOMMERCE
6287 FILE BIOSIS
127* FILE BIOTECHABS
127* FILE BIOTECHDS
3979* FILE BIOTECHNO
403 FILE CABA
1936 FILE CANCERLIT
7229 FILE CAPLUS
8* FILE CEABA-VTB
3* FILE CIN
53 FILE CONFSCI
5 FILE CROPB
3 FILE CROPU
800 FILE DISSABS
107 FILE DDFB
91 FILE DDFU
1329 FILE DGENE
107 FILE DRUGB
2 FILE INSTRUENWS
204 FILE DRUGU
2 FILE INRESREARH
128 FILE ENBAL
5214 FILE ENBASE
4341* FILE ESBIOBASE
575* FILE FEDRIP
0* FILE FOMAD
0* FILE FORGE
2* FILE FROSTI
5* FILE FSTA
464 FILE GENBANK
6 FILE HEALSAFE
110 FILE IFIPAT
102 FILE JICST-EPLUS
3* FILE KOSMET
3424 FILE LIFESCI
0* FILE MEDICONF
5940 FILE MEDLINE
60 FILE NIOSHTIC
70* FILE NTIS
0* FILE NUTFACEUT
2 FILE OCEAN
1031* FILE PASCAL
0* FILE PHARMVL
1 FILE PHIN
24 FILE PROMT
5900 FILE SCISEARCH
2647 FILE TOXCENTER
1414 FILE USPATFULL
62 FILE USPAT2
1 FILE VETU
107 FILE WPIDS
2 FILE WPIDV

L1 107 FILE WPINDEX
QUE CHROMATIN (P) BIND?

FILE 'BIOTECHNO, BIOSIS, LIFESCI, MEDLINE' ENTERED AT 13:38:58 ON 19 JUL 2004

L2 19630 S L1
L3 784 S L2 AND (ZINC (W) FINGER) OR ZFP
L4 14 S L3 AND INTRACELL?
L5 1608 S CHROMATIN AND BINDING (W) SITE AND PROTEIN
L6 842 DUP REM L5 (766 DUPLICATES REMOVED)
L7 39 S L6 AND ZINC (W) FINGER

=> s 17 not 14
L8 39 L7 NOT L4

=> s 18 and PY<=2000
3 FILES SEARCHED...

L9 15 L8 AND PY<=2000

=> d 19 1-15 bib ab

L9 ANSWER 1 OF 15 BIOTECHNO COPYRIGHT 2004 Elsevier Science B.V. on SIN
AN 2000:30808610 BIOTECHNO
TI Synthetic ***zinc*** ***finger*** transcription factor action at
an endogenous chromosomal site: Activation of the human erythropoietin
gene

AU Zhang L.; Spratt S.K.; Liu Q.; Johnstone B.; Qi H.; Raschke E.E.;

Jamieson A.C.; Rebar E.J.; Wolffe A.P.; Case C.C.

CS A.P. Wolffe, Sangamo Biosciences Inc., Point Richmond Tech. Center, 561

Canal Blvd., Richmond, CA 94804, United States.

E-mail: awolff@sangamo.com

SO Journal of Biological Chemistry, *** (27 OCT 2000)***, 275/43

(33850-33860), 39 reference(s)

CODEN: JBCHA3 ISSN: 0021-9258

DT Journal; Article

CY United States

LA English

SL English

AB We have targeted the activation of an endogenous chromosomal locus including the human erythropoietin gene using synthetic transcription factors. These transcription factors are targeted to particular DNA sequences in the 5'-flanking region of the erythropoietin gene through engineering of a ***zinc*** ***finger*** DNA binding domain. The DNA binding domain is linked to a VP16 transcriptional activation domain. We find that these synthetic transcription factors invariably activate transiently transfected templates in which sequences within the 5' flank of the erythropoietin gene are fused to a luciferase reporter. The efficiency of activation under these circumstances at a defined site is dependent on DNA binding affinity. In contrast, only a subset of these same ***zinc*** ***finger*** proteins is able to activate the endogenous chromosomal locus. The activity of these proteins is influenced by their capacity to gain access to their cognate elements within the ***chromatin*** infrastructure. ***zinc*** ***finger*** transcription factors will provide a powerful tool to probe the determinants of ***chromatin*** accessibility and remodeling within endogenous chromosomal loci.

L9 ANSWER 2 OF 15 BIOTECHNO COPYRIGHT 2004 Elsevier Science B.V. on STN
 AN 2000:3073681 BIOTECHNO
 TI An Ikaros-containing ***chromatin*** -remodeling complex in adult-type erythroid cells
 AU O'Neill D.W.; Schoetz S.S.; Lopez R.A.; Castle M.; Rabinowitz L.; Shor E.; Krawchuk D.; Goll M.G.; Renz M.; Seelig H.-P.; Han S.; Seong R.H.; Park S.D.; Agaloti T.; Munshi N.; Thanos D.; Erdjument-Bromage H.; Tempst P.; Bank A.
 CS A. Bank, Dept. of Genetics and Development, Hamner Health Sciences, 701 West 168th Street, New York, NY 10032, United States.
 E-mail: bank@ccu.ccc.columbia.edu
 MO Molecular and Cellular Biology, (***2000***), 20/20 (7572-7582), 59 references(s)
 CODEN: MCEBD4 ISSN: 0270-7306
 DT Journal; Article
 CY United States
 LA English
 SL English
 AB We have previously described a SWI/SNF-related ***protein*** complex (PYR complex) that is restricted to definitive (adult-type) hematopoietic cells and that specifically binds DNA sequences containing long stretches of pyrimidines. Deletion of an intergenic DNA- ***binding*** site for this complex from a human .beta.-globin locus construct results in delayed human .gamma.- to .beta.-globin switching in transgenic mice, suggesting that the PYR complex acts to facilitate the switch. We now show that PYR complex BNA-binding activity also copurifies with subunits of a second type of ***chromatin*** -remodeling complex, nucleosome-remodeling deacetylase (NuRD), that has been shown to have both nucleosome-remodeling and histone deacetylase activities. Gel supershift assays using antibodies to the ATPase-helicase subunit of the NuRD complex, Mi-2 (CHD4), confirm that Mi-2 is a component of the PYR complex. In addition, we show that the hematopoietic cell-restricted ***zinc*** ***finger*** ***protein*** Ikaros copurifies with PYR complex DNA-binding activity and that antibodies to Ikaros also supershift the complex. We also show that NuRD and SWI/SNF components coimmunopurify with each other as well as with Ikaros. Competition gel shift experiments using partially purified PYR complex and recombinant Ikaros ***protein*** indicate that Ikaros functions as a DNA-binding subunit of the PYR complex. Our results suggest that Ikaros targets two types of ***chromatin*** -remodeling factors - activators (SWI/SNF) and repressors (NuRD) - in a single complex (PYR complex) to the .beta.-globin locus in adult erythroid cells. At the time of the switch from fetal to adult globin production, the PYR complex is assembled and may function to repress .gamma.-globin gene expression and facilitate .beta.- to .beta.-globin switching.

L9 ANSWER 3 OF 15 BIOTECHNO COPYRIGHT 2004 Elsevier Science B.V. on STN
 AN 2000:30710842 BIOTECHNO
 TI Transcriptional activation by the PHD finger is inhibited through an adjacent leucine zipper that binds 14-3-3 proteins
 AU Halbach T.; Scheer N.; Werr W.
 CS W. Werr, Institut für Entwicklungsbiologie, Universität zu Köln, Gyrhofstrabe 17, 50923 Köln, Germany.
 E-mail: w.werr@uni-koeln.de
 MO Nucleic Acids Research, *** (15 SEP 2000)*** , 28/18 (3542-3550), 50 references(s)

CODEN: NARHAD ISSN: 0305-1048
 DT Journal; Article
 CY United Kingdom
 LA English
 SL English
 AB The PHD finger, a Cys.sub.4-His-Cys.sub.3 ***zinc*** ***finger***, is found in many regulatory proteins from plants or animals which are frequently associated with ***chromatin*** -mediated transcriptional regulation. We show here that the PHD finger activates transcription in yeast, plant and animal cells. In plant homeodomain transcription factors the PHD finger is combined with an upstream leucine zipper. Both domains together form a highly conserved 180 amino acid region called the ZIP/PHD motif and transcriptional activity of the PHD finger is masked when embedded in this motif. Our results indicate that the ZIP/PHD domain is a potential regulatory domain of PHD-HD proteins. The leucine zipper upstream of the PHD finger interacts with 14-3-3GF14.mu. from Arabidopsis thaliana and 14-3-3GF14-12 from maize via a leucine zipper conserved in helix 4 of various 14-3-3 proteins from plants and animals. PHD-type plant homeodomain proteins consequently may represent potential targets of 14-3-3 signalling.

L9 ANSWER 4 OF 15 BIOTECHNO COPYRIGHT 2004 Elsevier Science B.V. on STN
 AN 2000:30329448 BIOTECHNO
 TI Maternal-specific footprints at putative CTCF sites in the H19 imprinting control region give evidence for insulator function
 AU Szabo P.E.; Tang S.-H.E.; Rentsendorj A.; Pfeifer G.P.; Mann J.R.
 CS P.E. Szabo, Division of Biology, Research Inst. of the City of Hope, 1450 East Duarte Road, Duarte, CA 91010-3011, United States.
 E-mail: pszabo@coh.org
 MO Current Biology, *** (18 MAY 2000)*** , 10/10 (607-610), 18 references(s)
 CODEN: CUSLE2 ISSN: 0960-9822
 DT Journal; Article
 CY United Kingdom
 LA English
 SL English
 AB Parent-of-origin-specific expression of the mouse insulin-like growth factor 2 (Igf2) gene and the closely linked H19 gene are regulated by an intervening 2 Kb imprinting control region (ICR), which displays parent-specific differential DNA methylation [1,2]. Four 21 bp repeats are embedded within the ICR and are conserved in the putative ICR of human and rat Igf2 and H19, suggesting that the repeats have a function [3,4]. Here, we report that prominent DNA footprints were found in vivo on the unmethylated maternal ICR at all four 21 bp repeats, demonstrating the presence of ***protein*** binding. The methylated paternal ICR displayed no footprints. Significantly, the maternal-specific footprints were localized to putative binding sites for CTCF, a highly conserved ***zinc*** - ***finger*** DNA-binding ***protein*** with multiple roles in gene regulation including that of ***chromatin*** insulator function [5,6]. These results strongly suggest that the maternal ICR functions as an insulator element in regulating mutually exclusive expression of Igf2 and H19 in cis.

L9 ANSWER 5 OF 15 BIOTECHNO COPYRIGHT 2004 Elsevier Science B.V. on STN
 AN 1998:2B483729 BIOTECHNO
 TI Crystal structure of the BTB domain from PLZF

Ahmad K.F.; Engel C.K.; Prive G.G.
 G.G. Prive, Div. of Molecular/Structural Biol., Ontario Cancer Institute,
 University of Toronto, 610 University Avenue, Toronto, Ont. M5G 2M9,
 Canada.
 E-mail: prive@oci.utoronto.ca
 Proceedings of the National Academy of Sciences of the United States of
 America, *** (13 OCT 1998)***, 95/21 (12123-12128), 38 reference(s)
 CODEN: PNASAE ISSN: 0027-8424
 Journal; Conference Article
 United States
 English
 LA
 English
 SL
 AB
 The B7B domain (also known as the POZ domain) is an evolutionarily
 conserved ***protein*** - ***protein*** interaction motif found at
 the N terminus of 5-10% of C.sub.2H.sub.2-type ***zinc*** -
 finger transcription factors, as well as in some actin-
 associated proteins bearing the kelch motif. Many B7B proteins are
 transcriptional regulators that mediate gene expression through the
 control of ***chromatin*** conformation. In the human promyelocytic
 leukemia ***zinc*** ***finger*** (PLZF) ***protein***, the
 B7B domain has transcriptional repression activity, directs the
 protein to a nuclear punctate pattern, and interacts with
 components of the histone deacetylase complex. The association of the
 PLZF B7B domain with the histone deacetylase complex provides a mechanism
 of linking the transcription factor with enzymatic activities that
 regulate ***chromatin*** conformation. The crystal structure of the
 B7B domain of PLZF was determined at 1.9 .ANG. resolution and reveals a
 tightly intertwined dimer with an extensive hydrophobic interface.
 Approximately one-quarter of the monomer surface area is involved in the
 dimer intermolecular contact. These features are typical of obligate
 homodimers, and we expect the full-length PLZF ***protein*** to exist
 as a branched transcription factor with two C-terminal DNA-binding
 regions. A surface-exposed groove lined with conserved amino acids is
 formed at the dimer interface, suggestive of a peptide- ***binding***
 site. This groove may represent the site of interaction of the
 PLZF B7B domain with nuclear corepressors or other nuclear proteins.

number of ***zinc*** ***finger*** proteins, including the human
 BCL-6 ***protein***. By using a ***binding*** ***site***
 selection technique (CAST), a high affinity ***binding*** ***site***
 of the ***protein*** was determined to be
 (A/C)ACATCTG(G/T)(A/C), containing the E box core sequence motif. The
 protein was shown to repress transcription from a promoter
 linked to its target sequences and was hence named RP58 (Repressor
 protein with a predicted molecular mass of 59 kDa). Immunogold
 electron microscopic study revealed that almost all RP58 is localized in
 condensed ***chromatin*** regions. These observations demonstrate for
 the first time that a ***protein*** mediating a sequence-specific
 transcriptional repression associates with highly condensed
 chromatin. We suggest that RP58 may be involved in a molecular
 link between sequence-specific transcriptional repression and the
 organization of chromosomes in the nucleus.

L9 ANSWER 7 OF 15 BIOTECHNO COPYRIGHT 2004 Elsevier Science B.V. on STN
 AN 1998:28335589 BIOTECHNO
 TI TIF1.alpha.: A possible link between KRAB ***zinc*** ***finger***
 proteins and nuclear receptors
 AU Le Douarin B.; You J.; Nielsen A.L.; Chambon P.; Lessou R.
 CS P. Chambon, Inst. Genet./Biol. Molec./Cellulaire, CNRS/INSERM/ULP,
 Collège de France, BP 163, 67404 Illkirch Cedex, France.
 SO Journal of Steroid Biochemistry and Molecular Biology, (***1998***),
 65/1-6 (43-50), 35 reference(s)
 CODEN: JSBHEZ ISSN: 0960-0760
 S0960076097001751
 PUI Journal; Conference Article
 DT United Kingdom
 CY English
 LA English
 SL English
 AB Ligand-induced gene activation by nuclear receptors (NRs) is thought to
 be mediated by transcriptional intermediary factors (TIFs), that interact
 with their ligand-dependent AF-2 activating domain. Included in the group
 of the putative AF-2 Tifs identified so far is TIF1.alpha., a member of a
 new family of proteins which contains an N-terminal RBC (RING finger-B
 boxes-coiled coil) motif and a C-terminal bromodomain preceded by a PHD
 finger. In addition to these conserved domains present in a number of
 transcriptional regulatory proteins, TIF1.alpha. was found to contain
 several ***protein*** - ***protein*** interaction sites. Of these,
 one specifically interacts with NRs bound to their agonistic ligand and
 not with NR mutants that are defective in the AF-2 activity. Immediately
 adjacent to this 'NR box', TIF1.alpha. contains an interaction site for
 members of the ***chromatin*** organization modifier (chromo) family,
 HPI1.alpha. and MOD1, which both are heterochromatic proteins. Finally,
 TIF1.alpha. also has a ***binding*** ***site*** for KRAB
 silencing domains of C.sub.2H.sub.2 ***zinc*** ***finger***
 proteins. TIF1.beta., another member of the TIF1 gene family, has some
 interacting partners in common with TIF1.alpha.. TIF1.beta. can interact
 with HPI1.alpha., MOD1 and KRAB domains, but apparently not with NRs. Both
 TIF1.alpha. and TIF1.beta. repress transcription when fused to a DNA
 binding domain in transiently transfected mammalian cells. A model
 discussing the potential function(s) of TIFs in the control of
 transcription at the level of the ***chromatin*** template will be
 presented.

L9 ANSWER 6 OF 15 BIOTECHNO COPYRIGHT 2004 Elsevier Science B.V. on STN
 AN 1998:28471685 BIOTECHNO
 TI RP58 associates with condensed ***chromatin*** and mediates a
 sequence-specific transcriptional repression
 AU Aoki K.; Meng G.; Suzuki K.; Takashi T.; Kameoka Y.; Nakahara K.; Ishida
 R.; Kasai M.
 CS Toyama, Shinjuku-ku, Tokyo 162, Japan.
 E-mail: masataka@nih.go.jp
 SO Journal of Biological Chemistry, *** (09 OCT 1998)***, 273/41
 (26698-26704), 38 reference(s)
 CODEN: JBCHA3 ISSN: 0021-9258
 DT Journal; Article
 CY United States
 LA English
 SL English
 AB An approximately 120-amino acid domain present generally at the NH.sub.2
 termini, termed the POZ domain, is highly conserved in various proteins
 with ***zinc*** ***finger*** DNA binding motifs. We have isolated
 a novel ***protein*** sharing homology with the POZ domain of a

L9 ANSWER 8 OF 15 BIOTECHNO COPYRIGHT 2004 Elsevier Science B.V. on STN
AN 1998:28099683 BIOTECHNO
TI Fusion proteins of the retinoic acid receptor-.alpha. recruit histone
AU deacetylase in promyelocytic leukaemia
M.; Fanelli M.; Rutaradt M.; Ferrara F.F.; Zanir I.; Seiser C.; Grignani
F.; Lazar M.A.; Minucci S.; Pelicci P.G.
CS P.G. Pelicci, Ist. Med. Int. Scienze Oncologiche, Perugia University,
06100 Perugia, Italy.
E-mail: ppelicci@iso.cilea.it
SO Nature, *** (19 FEB 1998)***, 391/6669 (815-818), 29 reference(s)
DT CODEN: NATUAS ISSN: 0028-0836
CY Journal: Article
LA United Kingdom
SL English
AB The transforming proteins of acute promyelocytic leukaemias (APL) are
fusions of the promyelocytic leukaemia (PML) and the promyelocytic
leukaemia ***zinc*** - ***finger*** (PLZF) proteins with retinoic
acid receptor-.alpha. (RAR.alpha.). These proteins retain the RAR.alpha.
DNA and retinoic acid (RA)-binding domains, and their ability to block
haematopoietic differentiation depends on the RAR.alpha. DNA-binding
domain. Thus RA-target genes are downstream effectors. However, treatment
with RA induces differentiation of leukaemic blast cells and disease
remission in PML-RAR.alpha. APLs, whereas PLZF-RAR.alpha. APLs are
resistant to RA. Transcriptional regulation by RARs involves
modifications of ***chromatin*** by histone deacetylases, which are
recruited to RA-target genes by nuclear co-repressors. Here we show that
both PML-RAR.alpha. and PLZF-RAR.alpha. fusion proteins recruit the
nuclear co-repressor (N-CoR)-histone deacetylase complex through the
RAR.alpha. CoR box. PLZF-RAR.alpha. contains a second, RA-resistant
binding ***site*** in the PLZF amino-terminal region. High
doses of RA release histone deacetylase activity from PML-RAR.alpha., but
not from PLZF-RAR.alpha.. Mutation of the N-CoR ***binding***
site abolishes the ability of PML-RAR.alpha. to block
differentiation, whereas inhibition of histone deacetylase activity
switches the transcriptional and biological effects of PLZF-RAR.alpha.
from being an inhibitor to an activator of the RA signalling pathway.
Therefore, recruitment of histone deacetylase is crucial to the
transforming potential of APL fusion proteins, and the different effects
of RA on the stability of the PML-RAR.alpha. and PLZF-RAR.alpha.
co-repressor complexes determines the differential response of APLs to
RA.

L9 ANSWER 9 OF 15 BIOTECHNO COPYRIGHT 2004 Elsevier Science B.V. on STN
AN 1995:25265905 BIOTECHNO
TI A drosophila ***protein*** that imparts directionality on a
variegation
AU ***chromatin*** insulator is an enhancer of position-effect
CS Gerasimova T.I.; Gdula D.A.; Gerasimov D.V.; Simonova O.; Corces V.G.
Department of Biology, Johns Hopkins University, Baltimore, MD 21218,
United States.
SO Cell, (***1995***), 82/4 (587-597)
DT CODEN: CELL25 ISSN: 0092-8674
CY Journal: Article
LA United States
SL English
AB The suppressor of Hairy wing (su(Hw)) ***protein*** inhibits the
function of transcriptional enhancers located distally from the promoter
with respect to the location of su(Hw)-binding sites. This polarity is
due to the ability of the su(Hw)-binding region to form a
chromatin insulator. Mutations in modifier of mdg4 (mod(mdg4))
enhance the effect of su(Hw) by inhibiting the function of enhancers
located on both sides of the su(Hw)-binding region. This inhibition
results in a variegated expression pattern, and mutations in mod(mdg4) act
as classical enhancers of position-effect variegation. The mod(mdg4) and
su(Hw) proteins interact with each other. The mod(mdg4) ***protein***
controls the nature of the repressive effect of su(Hw): in the absence of
mod(mdg4) ***protein***, su(Hw) exerts a bidirectional silencing
effect, whereas in the presence of mod(mdg4), the silencing effect is
transformed into unidirectional repression.

L9 ANSWER 10 OF 15 BIOTECHNO COPYRIGHT 2004 Elsevier Science B.V. on STN
AN 1994:24337786 BIOTECHNO
TI Requirement for RGR1 and SIN4 in RME1-dependent repression in
AU Saccharomyces cerevisiae
AU Covitz P.A.; Song W.; Mitchell A.P.
CS Department of Biological Sciences, Stanford University, Stanford, CA
94305, United States.
SO Genetics, (***1994***), 138/3 (577-586)
DT CODEN: GENTAJ ISSN: 0016-6731
CY Journal: Article
LA United States
SL English
AB RME1 is a ***zinc*** - ***finger*** ***protein*** homolog that
functions as a repressor of the meiotic activator IME1:RME1 is unusual
among yeast repressors in two respects: it acts over a considerable
distance (2 kbp) and it can activate transcription from a ***binding***
site separated from its natural flanking region. To identify
genes required for RME1 to exert repression, we have selected mutants
with improved RME1-dependent activation. One rare mutant was defective in
RME1-dependent repression of an artificial reporter gene as well as the
native IME1 gene. The mutation permits sporulation of a/a diploids, which
express RME1 from its natural promoter, and of a/a.alpha. diploids
constructed to express RME1 from the GAL1 promoter. The mutation also
causes temperature-sensitive growth and a methionine or cysteine
requirement. Analysis of a complementing genomic clone indicates that the
mutation lies in a known essential gene, RGR1. Prior studies have
indicated a functional relationship between RGR1 and SIN4 (also called
TSF3); we have found that a sin4 null mutation also causes a defect in
RME1-dependent repression and a methionine or cysteine requirement. The
rgr1 and sin4 mutations do not cause a reduction of RME1 polypeptide
levels. The defect in RME1-dependent repression may result from effects
of sin4 and, presumably, rgr1 on ***chromatin*** structure.

L9 ANSWER 11 OF 15 BIOTECHNO COPYRIGHT 2004 Elsevier Science B.V. on STN
AN 1992:22102723 BIOTECHNO
TI The polyhomeotic gene of Drosophila encodes a ***chromatin***
protein that shares polytene chromosome-binding sites with
Polycamb
AU DeCamillis M.; Cheng N.; Pierre D.; Brock H.W.
CS Department of Zoology, University of British Columbia, Vancouver, BC V6T

myeloid precursors. Previous work has also shown that PLZF can recruit components of the nuclear receptor co-repressor complexes, such as N-CoR and histone deacetylase (HDAC) through the POZ domain, raising the possibility that effects of PLZF on gene transcription are mediated through an HDAC dependent mechanism. We now show directly that transcriptional repression by PLZF correlates with deacetylation of core histones present in ***chromatin*** surrounding its DNA ***binding*** ***site***. Furthermore, the ability of PLZF to repress transcription and cause deacetylation of histone H4 in vivo is highly dependent on acetylation of lysine residues in its C-terminal. ***zinc*** - ***finger***. Acetylation of PLZF is mediated by acetyltransferase activity of the p300 ***protein*** (but not SMC-1 or p/CAF), with which PLZF associates in vitro and in vivo. As expected from the above results, transient co-expression of p300, but not its histone acetyltransferase domain deficient mutant, enhances transcriptional repression by PLZF. Mutating the target lysine residues to arginines, which cannot be acetylated, completely abolishes the ability of PLZF to cause histone deacetylation and repress transcription in vivo. Furthermore, co-expression of HDAC3 (but not HDAC1), which also interacts with and deacetylates PLZF in vitro, relieves repression by the wild type PLZF ***protein***. Taken together, our results indicate that a histone deacetylase dependent transcriptional repressor, can be regulated through a cycle of acetylation/deacetylation of lysine residues in its most C-terminal ***zinc*** - ***finger***. The indirect effect of acetylation and deacetylation of PLZF thus serves to stimulate and inhibit its ability to repress target gene transcription.

124, Canada.
Genes and Development, (***1992***), 6/2 (223-232)
CODEN: GDEP; ISSN: 0950-9369
Journal; Article
United States
English
SL
The Polycomb group (PcG) genes in Drosophila melanogaster are required for maintenance of correct spatial expression of homeotic genes, and their products are thought to form either a regulatory network or act as a multimeric complex. Recently, it has been suggested that because of homology between Polycomb (Pc) and Su(Hw)205, PcG genes encode ***chromatin*** proteins required for the maintenance of a determined state in ***chromatin***. The polyhomeotic (ph) gene is a member of the PcG of genes. We present DNA sequence of a ph cDNA, which encodes a 169-kD ***protein*** with a single putative ***zinc*** - ***finger***, a serine/threonine-rich region, and has glutamine repeats, suggesting that ph is a DNA-binding ***protein***. Polyclonal antisera directed against ph ***protein*** bind to sim.80 sites on polytene chromosomes. Most of these sites appear to be the same as those recognized by antibodies to Pc ***protein***. ph ***protein*** binds to insertion sites of constructs containing DNA from the bithoraxoid (bxd) region of the Bithorax complex, showing that ph binding to ***chromatin*** is DNA dependent. The same bxd constructs are recognized by Pc ***protein***, strongly supporting the hypothesis that ph and Pc interact directly.

ANSWER 13 OF 15 BIOSIS COPYRIGHT 2004 BIOLOGICAL ABSTRACTS INC. on STN
AN 2001:312498 BIOSIS
DN PREV200100312498
TI Identification of the ***protein*** 4.2 gene as a direct target of the TAL1/SCL transcription factor in differentiating murine erythroleukemia cells.
AU Xu, Zhixiong [Reprint author]; Huang, Suming [Reprint author]; Chang, Long-Sheng; Brandt, Stephen J. [Reprint author]
CS Medicine, Vanderbilt University Medical Center, Nashville, TN, USA
SO Blood, (November 16, 2000) Vol. 96, No. 11 Part 1, pp. 497a. Print.
Meeting Info.: 42nd Annual Meeting of the American Society of Hematology, San Francisco, California, USA. December 01-05, 2000. American Society of Hematology.
CODEN: BLOOD. ISSN: 0006-4971.
Conference; (Meeting)
Conference; Abstract; (Meeting Abstract)
English
ED Entered STN: 27 Jun 2001
LA Last Updated on STN: 19 Feb 2002
AB The TAL1/SCL gene, originally identified through its involvement by a recurrent chromosomal translocation, encodes a basic helix-loop-helix (bHLH) transcription factor with an essential role in embryonic hematopoiesis. Although TAL1 likely alters the transcription of a specific set of genes, no targets have been definitively identified. ***Binding*** ***site*** selection assays in erythroid cells have suggested that TAL1 contributes to a multiprotein DNA-binding complex that also includes the ***zinc*** - ***finger*** ***protein*** (GATA-1) and has a preference for a bipartite sequence motif containing an E box (bHLH ***protein*** ***binding*** ***site***) and a GATA site. We identified two such E box-GATA elements in the proximal promoter of the

ANSWER 12 OF 15 BIOSIS COPYRIGHT 2004 BIOLOGICAL ABSTRACTS INC. on STN
AN 2001:313274 BIOSIS
DN PREV200100313274
TI Acetylation and deacetylation of the promyelocytic leukemia ***zinc*** - ***finger*** (PLZF) regulate its ability to function as a transcriptional repressor.
AU Guidez, Fabien [Reprint author]; Ivins, Sarah [Reprint author]; Owen, Gareth [Reprint author]; Rowe, Nicola [Reprint author]; Zelent, Arthur [Reprint author]
CS Leukemia Research Fund Center at the Institute of Cancer Research, Chester Beatty Laboratories, London, UK
SO Blood, (November 16, 2000) Vol. 96, No. 11 Part 1, pp. 453a. Print.
Meeting Info.: 42nd Annual Meeting of the American Society of Hematology, San Francisco, California, USA. December 01-05, 2000. American Society of Hematology.
CODEN: BLOOD. ISSN: 0006-4971.
Conference; (Meeting)
Conference; Abstract; (Meeting Abstract)
English
ED Entered STN: 4 Jul 2001
LA Last Updated on STN: 19 Feb 2002
AB The PLZF ***protein***, originally identified as a fusion with RARalpha in rare cases of all-trans-retinoic acid resistant acute promyelocytic leukemia, is a transcriptional repressor characterized by a C-terminal DNA-binding domain, consisting of nine Kruppel-like zinc fingers, and an N-terminal ***protein*** / ***protein*** interaction domain, the POZ domain. Expression studies of PLZF throughout hematopoiesis, as well as its over-expression in hematopoietic progenitor cells, suggest that in addition to being involved in leukemogenesis, PLZF plays an important role in regulating growth and differentiation of normal

murine ***protein*** 4.2 gene and investigated whether this gene could be a target for these transcription factors. First, a TAL1- and GATA-1-containing complex was detected by gel shift analysis using both E box-GATA elements in the promoter as probes. Further, an increase in these DNA-binding activities was observed with DMSO-induced differentiation of murine erythroleukemia (MEL) cells, concomitant with an increase in expression of endogenous ***protein*** 4.2 mRNA. Cold competitor studies and DNA-binding assays with mutated probes indicated the requirement for both E box and GATA sites in these elements for formation of these binding complexes. In addition, reporter assays showed that DMSO-induced promoter activity decreased by approximately 75% and 90%, respectively, with mutation of either E box or GATA site, suggesting that both elements contribute to promoter activity and that the E box and GATA sites in these elements are both required for maximal induction of ***protein*** 4.2 promoter activity during MEL cell differentiation.

An expression vector for TAL1 increased promoter activity in reporter assays when cotransfected with its DNA-binding partner E47, GATA-1, and the LIM domain ***protein*** LMO2. Finally, an increase in endogenous ***protein*** 4.2 gene expression and in E box-GATA DNA-binding activity was observed when TAL1 was overexpressed in cells, a decrease in both was observed when a binding-defective TAL1 dominant negative mutant was introduced, and direct evidence for TAL1 occupancy of the promoter in cells was obtained by ***chromatin*** immunoprecipitation analysis. In sum, these data establish the ***protein*** 4.2 gene as a physiologic target of a TAL1- and GATA-1-containing complex in differentiating murine erythroleukemia cells.

ANSWER 14 OF 15 LIFESCI COPYRIGHT 2004 CSA ON STN

L9 1999:1250 LIFESCI

AN TIF1 alpha : A possible link between KRAB ***zinc*** ***finger*** proteins and nuclear receptors

LA English

AB Ligand-induced gene activation by nuclear receptors (NRs) is thought to be mediated by transcriptional intermediary factors (TIFs), that interact with their ligand-dependent AF-2 activating domain. Included in the group of the putative AF-2 TIFs identified so far is Tif1 alpha, a member of a new family of proteins which contains an N-terminal RBCC (RING finger-B boxes-coiled coil) motif and a C-terminal bromodomain preceded by a PHD finger. In addition to these conserved domains present in a number of transcriptional regulatory proteins, Tif1 alpha was found to contain several ***protein*** - ***protein*** interaction sites. Of these, one specifically interacts with NRs bound to their agonistic ligand and not with NR mutants that are defective in the AF-2 activity. Immediately adjacent to this 'NR box', Tif1 alpha contains an interaction site for members of the ***chromatin*** organization modifier (chromo) family, HPI1 alpha and MOD1, which both are heterochromatinic proteins. Finally,

Tif1 alpha also has a ***binding*** ***site*** for KRAB silencing domains of C sub(2)H sub(2) ***zinc*** ***finger*** proteins. Tif1 beta, another member of the Tif1 gene family, has some interacting partners in common with Tif1 alpha. Tif1 beta can interact with HPI1 alpha, MOD1 and KRAB domains, but apparently not with NRs. Both Tif1 alpha and Tif1 beta repress transcription when fused to a DNA binding domain in transiently transfected mammalian cells. A model discussing the potential function(s) of TIFs in the control of transcription at the level of the ***chromatin*** template will be presented.

L9 ANSWER 15 OF 15 MEDLINE ON STN

AN 97185912 MEDLINE

DN Published ID: 9033593

TI The solution structure of a specific GAGA factor-DNA complex reveals a modular binding mode.

CM Comment in: Nat Struct Biol. 1997 Feb;4(2):87-9. PubMed ID: 9033591

AU Omichinski J G; Pedone P V; Felsenfeld G; Gronenborn A M; Clore G M

CS Laboratories of Chemical Physics, National Institute of Diabetes and Digestive and Kidney Diseases, National Institutes of Health, Bethesda, Maryland 20892-0520, USA.

SO Nature structural biology, *** (1997 Feb)*** 4 (2) 122-32.

CY United States

DT Journal; Article; (JOURNAL ARTICLE)

LA English

FS Priority Journals

EM 199703

ED Entered STN: 19970327

AB The structure of a complex between the DNA binding domain of the GAGA factor (GAGA-DBD) and an oligonucleotide containing its GAGAG consensus ***binding*** ***site*** has been determined by nuclear magnetic resonance spectroscopy. The GAGA-DBD comprises a single classical Cys2-His2 ***zinc*** ***finger*** core, and an N-terminal extension containing two highly basic regions, BR1 and BR2. The ***zinc*** ***finger*** core binds in the major groove and recognizes the first three GAG bases of the consensus in a manner similar to that seen in other classical ***zinc*** ***finger***-DNA complexes. Unlike the latter, which require tandem ***zinc*** ***finger*** repeats with a minimum of two units for high affinity binding, the GAGA-DBD makes use of only a single finger complemented by BR1 and BR2. BR2 forms a helix that interacts in the major groove recognizing the last G of the consensus, while BR1 wraps around the DNA in the minor groove and recognizes the A in the fourth position of the consensus. The implications of the structure of the GAGA-DBD-DNA complex for ***chromatin*** remodelling are discussed.

=> log h

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